Acute Acalculous Cholecystitis

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ACUTE acalculous cholecystitis is a diagnosis that is usually made at the time of operation and is sometimes erroneous because of an overlooked small calculus in the gallbladder. The condition is a serious one and fortunately relatively rare, but its incidence is actually increasing. We have made the diagnosis at operation in 139 patients among 2253 operated on for acute cholecystitis between September 1, 1932 and September 1, 1977 at The New York Hospital–Cornell Medical Center. The condition occurs in all ages ranging from very young to very old. The incidence is somewhat more frequent in males with the majority among those 65 years of age and older. It is associated with debilitated states such as seen in the elderly, and follows an unrelated surgical procedure and major trauma. The probability of making a preoperative differential diagnosis between calculous and acalculous acute cholecystitis at present is quite remote. Approximately one-half the patients were seriously ill when they were admitted to the hospital. Those who we treated surgically have a mortality rate of 6.5%. Because of their critical condition palliative procedures to ameliorate their precarious state are employed postponing a definitive procedure until their general condition has improved.

There are a number of factors related to the suggestive pathology of this condition, its clinical course and eventual recovery or death (Table 1). These are briefly discussed under those considered to be etiologic and those that have a bearing on the clinical course. Readily demonstrated were surgical operations unrelated to the biliary tract, massive trauma, burns, and postpartum instances following long and difficult labors were readily demonstrated. Of equal importance (was severe systemic disease of sufficient magnitude and duration occurring in all age groups including the very young but also equally dramatic in the elderly. Cardiovascular disease ranging from hypertension, arteriosclerosis to cardiac failure regardless, of etiology and sometimes associated with obesity, appeared to be of considerable clinical significance in almost half of the group reported.

We have repeatedly reviewed our clinical observations of our patients and the reports of others in seeking a common pattern of background for the genesis of acute acalculous cholecystitis. Massive trauma including burns and debilitating disease (particularly infections) is suggestive that a substance that effects the wall of the gallbladder may be liberated from breaking down tissue or infection. The observations of Mann[8] reported in 1921 from the Mayo Clinic describe the pathology in dogs following the intravenous injection of Dakin's solution and indicate the susceptibility of the gallbladder to a blood borne "noxious" substance. The possible role of "something" from injured tissue merits further investigation together with other accompanying distortion of normal physiological processes.

In addition to the 68 patients noted above in which predisposing factors related to acute acalculous cholecystitis were manifested there were 71 in which no such factor could be determined.

Patients with no Specific Factor Recognized as Related to Acute Acalculous Cholecystitis

A total of 71 patients (42 male; 29 female) ranging from age 11 to 80 were found to have no specific factor relating to acute acalculous cholecystitis at operation. Nineteen of these patients were 65 years old or older. It is probable that if we were to select an equal number of patients coming to surgery in this age range that we might demonstrate an equal number of comparable disabilities that might be related to their presenting clinical status. It should be emphasized that the various factors that are discussed as they relate to acute acalculous cholecystitis may be less specific than we have assumed.

Regardless of the etiologic factors, this clinical entity is often a critical surgical problem. The recognition of acute cholecystitis is most important. Complete

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Submitted for publication: July 6, 1978.

0003-4932/79/0400/0458 $00.90

TABLE 1. *Predisposing Factors Recognized in 68 Patients* with Acute Acalculous Cholecystitis*

	Total	Male/ Female	Age Range	65 Years or Older
Following surgery, trauma, postpartum unrelated to biliary tract	24	17/7	24–89	10
Previous operation on biliary tract	2	1/1	57–74	1
Previous history and recognition of liver disease (cirrhosis, pancreatitis)	8	5/3	32–82	4
Coexisting disease states: infection, bacteremia, septicemia	8	4/4	9–72	1
Cardiovascular disease	24	15/9	38–90	14
Hypertension	18			
Arteriosclerosis, myocardial failure	4			
Phlebitis	2			
Obesity	10	3/7	40–77	5

* Eight of the above 68 patients were considered in 2 categories, 4 females and 4 males; five of the 8 were 65 years of age or older.

evaluation, close and constant observation of the course, usually reveals indications for immediate surgical interruption of the pathological process. The nature and the extent of the surgical procedure depend upon the findings exposed at operation.

In 1947 we reported 17 patients who developed acute cholecystitis following surgical treatment of conditions unrelated to the biliary tract during a period of 14 years.[3] We made no comment about the differentiation between calculous and acalculous disease, implying that calculi played a dominant role in the genesis of acute cholecystitis under these circumstances. In a subsequent report[5] of 18 patients in this category operated upon during a period of nine years (1946–1955), 14 had cholelithiasis and in four no calculi were found in the gallbladder or ductal system. In the present series, of 139 patients with acute acalculous cholecystitis, 24 developed acute cholecystitis following surgery unrelated to the biliary tract, trauma, or postpartum.

Patients Developing Acute Acalculous Cholecystitis Following Surgery Unrelated to Biliary Tract, Trauma, Burns and Postpartum

Twenty-four patients developed acute acalculous cholecystitis following surgery unrelated to the biliary tract, trauma, burns or postpartum. Seventeen of these patients were male and seven were female, ranging in age from 24 to 89. Ten of the 24 patients were 65 years old or older.

Example Cases

Case 1. An 89-year-old woman (104 91 72) developed an acute cholecystitis 5 weeks after reduction and Jewett nailing for fracture of the hip. Cholecystostomy was done. No calculi were found. The patient made a satisfactory recovery and remained well until lost to follow-up.

Case 2. A 54-year-old man (104 08 59) developed an acute cholecystitis following an exploratory laparotomy and jejunostomy for intestinal obstruction. Cholecystostomy was done and no stones found. He had had some disability from an associated rheumatoid arthritis for an indefinite period. His recovery was satisfactory.

Case 3. A 24-year-old woman (28 80 14) underwent medical induction of labor followed by a normal delivery on the sixth day after admission. Labor was somewhat prolonged but otherwise not unusual. At four days postpartum she developed sudden onset of severe epigastric pain and manifestations of an acute cholecystitis. A cholecystectomy was done and no stones were found. Her recovery was satisfactory and she remained symptom free for a little over three years when she was lost to follow-up.

Patients Developing Acute Acalculous Cholecystitis in Whom Cardiovascular Disease Including Hypertension, Atherosclerosis, Myocardial Failure and Phlebitis were Believed to be of Etiological Significance

There were a total of 24 patients in this group, of which 15 were male and nine were female. The patients ranged in age from 38 to 90 years old. Fourteen patients were 65 years old or older.

Example Cases

Case 1. A 90-year-old man (105 33 15) with cardiovascular disease (including congestive heart failure) was found to have acute acalculous cholecystitis at operation. Cholecystectomy was performed as well as exploration of the common duct. The patient made a satisfactory recovery and was discharged on May 10, 1977, 20 days after operation.

Case 2. A 55-year-old man (111 27 56) with metastatic cancer of the lung in addition to cardiovascular disease with hypertension and cardiac enlargement was operated on for acute acalculous cholecystitis. Cholecystostomy was done. His postoperative course was without event and he was discharged 15 days after operation.

Case 3. A 40-year-old woman (48 02 90) was known to have had diabetes mellitus and hypertension for some time. She was operated on 12.16.60 for acute acalculous cholecystitis and a cholecystostomy was done. She made a satisfactory recovery. Eight months later the patient was readmitted and cholecystectomy was performed. No calculi were present. The patient made a good recovery and has re-

mained symptom free, as far as the biliary tract is concerned, for several years.

The substance of this paper rests on the two groups that have been divided according to the primary operation, those treated by cholecystectomy as a definitive procedure and those treated by cholecystostomy as a compromise measure.

Acute Acalculous Cholecystitis Treated by Cholecystectomy

The definitive procedure of cholecystectomy is the procedure of choice because it effectually relieves the presenting problem and largely eliminates future biliary tract disease. Unless there are contraindications, the most important being a limited capacity of the patient to withstand the burden of such an operation, cholecystectomy should be performed. This is best decided at the operation (except for those who during preoperative evaluation are found to be poor risks either because of their overall status or because of some specific impairment of organ or system such as a history of recent myocardial infarction[17]). Those patients subjected to cholecystectomy have, as a group, been younger and more robust than those who have had cholecystostomy.

Careful evaluation of the ductal system is mandatory for those undergoing cholecystectomy and if the surgeon feels that choledocholithiasis is probable there should be a common duct exploration. Among the 78 patients subjected to cholecystectomy, 5 had common duct exploration as well. Forty-six patients were male and 32 were female. The patients ranged from nine to 90 years of age; 16 of which were 65 years old or older. Of the 73 patients whose primary operation was cholecystectomy alone, one patient demonstrated a calculus in the common duct at a second procedure. Another patient with symptoms referable to the biliary tract was found to have a gallbladder remnant and a long cystic duct but no calculi at a second operation.

Deaths Following Cholecystectomy for Acute Acalculous Cholecystitis

Seventy-eight patients were treated by cholecystectomy. Five also had exploration of the common duct. There were 3 deaths, all males, aged 9, 56 and 65.

The 9-year-old boy had been treated for typhoid

TABLE 2. *Deaths Following Cholecystectomy and Cholecystostomy for Acute Acalculous Cholecystitis*

Hist. No.	Age	Sex	Year	Diagnosis	Related Factor	Complication	Day Postop.	Cause of Death	Autopsy
3 21 61*	9	M	1934	Gangrenous c perforation	Infection peritonitis	Wound infection peritonitis	9	Typhoid peritonitis	Yes
118 86 44*	56	M	1970	Gangrenous c perforation	23 days p.o. resection aortic aneurysm pancreatitis	Renal failure pancreatitis	2	Renal failure	Yes
100 50 15*	65	M	1973	Acute cholecystitis	42 days p.o. esophago-gastrectomy	Infection, abd. Phlebitis	36	Pulmonary embolus	No
5 18 96†	66	F	1935	Gangrenous c perforation	Hypertension	Cardiac failure peritonitis	2	Peritonitis	Yes (stone found)
49 81 47†	46	M	1948	Acute cholecystitis	Hypertension	Uremia acute hemorrhagic pancreatitis	5	Necrotizing arteritis pancreatitis	Yes
106 97 91†	65	F	1966	Emphysematous cholecystitis gangrene c perforation	Obesity	Multiple cardiac arrests	17	Pseudomonas septicemia	Yes
53 12 02†	70	M	1968	Acute cholecystitis	Laennec's cirrhosis alcoholism	Hepatic failure pneumonia	8	Acute renal tubular necrosis hepatic failure	Yes
103 25 34†	72	F	1968	Acute cholecystitis	Infection	Septicemia	25	Septicemia	No
128 24 19†	71	M	1972	Acute cholecystitis	24 hrs p.o. hip replacement	Septicemia	22	Peritonitis perforation of cecum	Yes

* Cholecystectomy performed.
† Cholecystostomy performed.

fever in the hospital for a period of 21 dyas. He suddenly developed an acute abdomen believed to be secondary to a perforation of the intestine. At operation a perforated gangrenous gallbladder was removed. The patient died nine days later. Autopsy demonstrated typhoid infection and peritonitis.

The other two patients developed an acute cholecystitis following prolonged operative procedures. A 56-year-old man developed an acute cholecystitis 23 days after resection of an aortic aneurysm while in renal failure. A gangrenous fundus of the gallbladder with a perforation was found. Cholecystectomy was done and death occurred 2 days later. At postmortem pancreatitis was present. Death was probably due to renal failure.

The third patient, a 65-year-old man, had undergone esophagogastrectomy. His postoperative course was difficult and 45 days after surgery he developed unequivocal signs of an acute cholecystitis. Cholecystectomy was done and no calculi were identified. During the next 36 days the patient failed to do well, he died suddenly of a probable pulmonary embolus.

Cholecystostomy for Acute Acalculous Cholecystitis

Sixty-one patients (37 male; 24 female) had cholecystostomy instead of cholecystectomy because of contraindications. As a group, the patients were older (age range: 11–89), much less robust and, in the opinion of the surgeon, possessed a potential handicap so far as postoperative complications and possible death. A cholecystostomy done under local anesthesia is a procedure of minimal burden that provides decompression of the ductal system and prevents perforation of the gallbladder where it has not already occurred. Over half of these individuals were 65 years of age or more. The extent of the disease and prevalence of impaired organ or system function accounted for the six deaths that occurred in these patients rather than the procedure.

Deaths Following Cholecystostomy for Acute Acalculous Cholecystitis

There were six deaths following primary cholecystostomy for acute acalculous cholecystitis (3 males and 3 females). Five of the six were 65 years of age or older. Systemic infection and organ or system failure were major factors in the terminal course following operation.

Cholecystostomy was performed rather than cholecystectomy because of the precarious condition of the patient at the time of operation.[2] The lesser procedure imposed a minimal burden upon the patient's capacity to tolerate the burden of surgery.

Secondary Operations

Diagnosis of acute acalculous cholecystitis is made at operation and is a presumptive diagnosis in our experience. Twenty-five of the 139 patients operated on were later found to have calcareous material within the gallbladder or ductal system. Only one of the 25 patients underwent cholecystectomy as a primary procedure. One additional patient treated by cholecystostomy was found at autopsy to have a small calculus in the cystic duct proximal to the wall of the gallbladder (Fig. 1.).

Of 61 patients having cholecystostomy as a primary procedure, 24 had calcareous material removed at a later operation. Twenty-one of the 61 subsequently had a cholecystectomy, with calcareous material recovered in 12. Thirteen patients were later subjected to cholecystectomy and common duct exploration with calcareous material removed from the gallbladder in four and from the common duct in one. Calculi were present in both the gallbladder and the common duct in three. Three patients had a secondary cholecystostomy for acute cholecystitis with recovery of stones in one 16 months after the primary cholecystostomy.

A Malecot or a mushroom catheter was placed in the gallbladder and used to maintain the decompression for ten days or more. These catheters are foreign bodies and cholesterol crystals from the bile are believed to precipitate on their surface. Because trauma to the mucosa with some hemorrhage, sludge or amorphous-like material often forms and because it resembles calcareous material a diagnosis of calculi is made.

One patient following cholecystostomy was subjected to a choledochotomy only because it was felt that the common duct was larger than normal. A marked degree of edema in and around the triangle of Calot led the surgeon to believe cholecystectomy would be extremely hazardous. A stone was recovered from the common duct.

It is believed that calcareous or amorphous material forming in the gallbladder can readily pass into the common duct and be retained in it. One can only hypothesize on this but it is suggested that these findings resulted from response to a foreign body, the catheter in the gallbladder. In the same respect, calculous biliary tract disease may develop following cholecystostomy.

Deaths Following Secondary Procedure on Biliary Tract

The two deaths that followed a secondary procedure are of significance in the overall management of patients who have had a cholecystostomy as a primary operation. A 68-year-old woman, refused to have a cholecystectomy following cholecystostomy as the

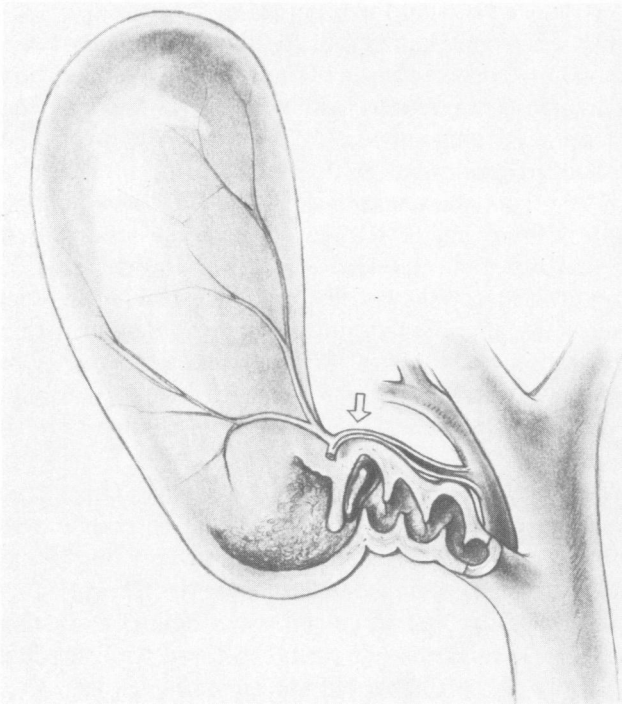

FIG. 1a. A 66-year-old woman with a gangrenous fundus of the gallbladder and perforation. At operation diagnosis was acute acalculous cholecystitis. Death occurred two days later. Autopsy revealed a small triangular calculus in the adjacent first valve of the cystic suct.

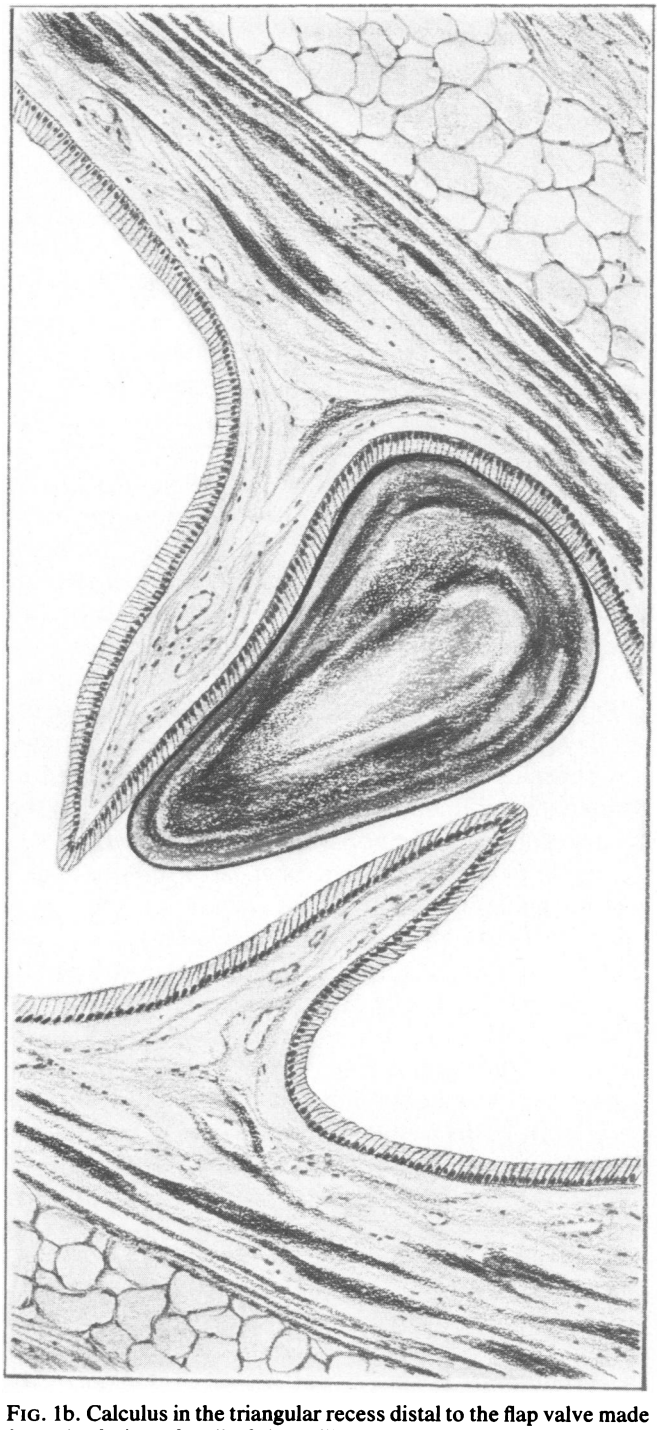

FIG. 1b. Calculus in the triangular recess distal to the flap valve made from the fusion of wall of the gallbladder and cystic duct.

primary operation for acute acalculous cholecystitis in 1960 because she remained symptom free. Seven years later at age 75 she developed acute cholecystitis. A secondary cholecystostomy was done in which no calculi were found. Six days later she had a fatal pulmonary embolus secondary to phlebitis.

Another patient (57-year-old man), following cholecystostomy for acute acalculous cholecystitis, continued to have a low grade fever and loss of bile through the cholecystostomy tube suggesting common duct obstruction. At a second operation a defect said to be secondary to necrosis of the wall of the common duct was identified. A cholecystectomy was done, the common duct explored and a T tube inserted for decompression. Stones were recovered from the gallbladder. Death occurred 11 days later. No autopsy was performed but death was attributed to peritonitis.

A third operation was performed on 3 patients, a cholecystectomy with calcareous material in the gallbladder in one; a cholecystectomy and common duct exploration in another with well formed stones removed from both the gallbladder and the common duct; and choledochotomy with a calculus recovered in the third.

Thus in 38 of 61 patients treated primarily by cholecystostomy, and always with drainage of the gallbladder by a self-retaining catheter, calcareous like material was recovered later, in 24 patients, at a secondary operation from either the gallbladder or ductal system. One patient had calculi removed during a second chole-

cystostomy for acute cholecystitis. Three months later cholecystectomy and common duct exploration was performed with stones removed from both the gallbladder and the common duct.

Numerous investigators have attempted to produce acute cholecystitis in animals with varying degrees of success. Directing the external secretion of the pancreas into the gallbladder readily produced an acute reaction of the gallbladder.[18] This was clinically demonstrated by Opie in 1901.[11] It is, however, one of several possible causes of acute cholecystitis. A review of investigations related to the experimental production of acute cholecystitis in animals (cats and dogs) using substances that might produce an inflammatory reaction in the gallbladder suggest a toxic factor delivered by the blood stream.

The most striking results using such an approach were reported by Mann[8] from the Mayo Clinic in 1921. By the intravenous injection of a solution of chlorinated soda he produced the definite reaction of breaking down the capillaries and infiltrating of the wall of the gallbladder with blood which he produced by introducing 5 cc/kg of Dakin's solution intravenously in dogs. Although they thought that chlorine was probably the specific substance in the solution it was eventually concluded that chlorine alone was not the cause.

Murphy[10] in 1930 used the same approach to produce changes in the gallbladder wall in a study relative to the emptying of the gallbladder. He employed a substance, eusol, containing sodium hypochlorite which was introduced intravenously. The degree of reaction in the gallbladder was dependent on the dosage. In only one of ten animals did the gallbladder empty following administration of egg yolk and cream. All had sufficient inflammatory evidence to be considered as unequivocal acute cholecystitis.

They held that the solution reached the gallbladder through the blood stream and that there were gross changes in the gallbladder within two hours after the injection. He was searching for a toxic substance as a cause of acute cholecystitis.

These experiments are cited because of a possible relationship to acute acalculous cholecystitis to trauma, including surgical operations as well as severe generalized infection (septicemia). They suggest "a toxic factor" in this type of acute cholecystitis.

Mann[8] was able to observe changes in the gallbladder of a gross nature that began as early as one-half hour after injection of the solution in the anesthetized dog. In some of the animals the cystic duct was securely ligated and changes continued to take place which were comparable to those with a patent cystic duct. He felt that this proved conclusively that the changes were due to a substance brought to the gallbladder wall by the blood stream. At the end of 24 hours following injection the gallbladder was intensely inflamed, being most marked at the fundus of the gallbladder and extending over the entire exposed surface. He observed also that the lymphatics of the gallbladder became very prominent and were soon colored with the blood contained within them. The organ was distended with minimal edema. To him the common and hepatic ducts did not seem involved in the reaction.

Microscopic evaluation showed that the acutely inflamed gallbladder was characterized by rupture of the capillaries with cellular infiltration of the entire wall. Mucosal ulceration was widespread with scattered areas of necrosis in the submucosa. There was also hemorrhage into the lumen.

Sequential observations showed that the acute inflammatory reaction may last for several weeks during which time the gallbladder remained distended. There was minimal edema followed by slow improvement. Within three months the process had subsided to such an extent that the gallbladder appeared normal.

Pathology

We have been unable to determine any difference in the clinical manifestations between the acute calculous and acute acalculous cholecystitis. At operation the gross appearance of the gallbladder is quite similar. On palpation calculi may be palpable if present, but often they escape detection. The gallbladder without calculi as exposed at operation presents all phases of an acute inflammatory process ranging from edema of the wall, hydrops of the gallbladder, to marked thickening and induration and on to ischemic necrosis and gangrene with perforation. The markedly edematous gallbladder wall, as a fixed specimen when examined a few hours later may appear to be almost normal on microscopic examination. This is the early stage of acute cholecystitis; somewhat later ulceration of the mucosa, cellular infiltration of muscularis and serosa will be observed. A perforation visible on gross examination is usually evident at the junction of viable and nonviable tissue. Very often a search for bacteria is unrewarding but at times single bacteria and clusters of varied organisms may be recognized. Equally variable is the number of white cells, in some instances none can be found in an edematous wall. The gross and microscopic examination of the wall of an acute gallbladder of acute acalculous cholecystitis is indistinguishable from acute cholecystitis in the gallbladder with calculi or calculi in the cystic duct.[1] Usually we recognize the extent of the changes but seldom we do not recognize any changes

that might indicate the cause. In our search for the etiological factors in acute acalculous cholecystitis we are aware of the clinical events preceding the onset of the acute process in many patients. The two most frequently reported are, first, those following surgery or severe trauma unrelated to the biliary tract and second, in individuals with significant cardiovascular disease.

Etiology

From numerous published reports on acute acalculous cholecystitis and a survey of our experience with 139 patients, it is clear that there are many factors that one may suspect as being of etiological relationship.[6,7,12–15] However no single group of circumstances has been identified that may be said to indicate the genesis of acute acalculous cholecystitis.

Interesting observations of changes in the gallbladder of dogs, as reported by Mann[8] and Murphy[10] resemble somewhat those seen in clinical material. The effectual chemical substance and its exact mechanism is not evident. Were this to be accomplished it would be a noteworthy contribution.

Acute cholecystitis following surgical procedures for conditions unrelated to the biliary tract and massive trauma including burns has led to much discussion of the role of hypotension. Indeed in the young and robust, particularly adult males the relationship appears striking. It also seems equally applicable to the elderly patients with cardiovascular disease involving the arterial vessels supplying the gallbladder. In 1963 we published[4] in some detail the mechanism thought to be a plausible explanation for fulminating acute cholecystitis with perforation. The association of bile heavily ladened with colon bacilli can readily be envoked as being of importance in conjunction with stasis in the gallbladder regardless of the cause of the stasis. The overwhelming infection described above in a 9-year-old boy with typhoid fever emphasizes the potential role of a bacteremia regardless of the nature of the organism or its site of origin.

The normal physiology of the biliary system in a state of health begins with formation of bile within the liver and its passing to the gallbladder where in the intradigestive period it is concentrated to a 10:1 ratio. The passage of stomach contents in the duodenum results in the gallbladder emptying bile into the common duct and then into the duodenum. A disturbance in this highly coordinated process may provide circumstances that could result in a vicious cycle. Spasm of the sphincter of Oddi may result in an abnormal increase of pressure in the ductal system and gallbladder followed by regurgitation of pancreatic juice into the gallbladder causing an intense inflammatory reaction of the gallbladder mucosa. Increased pressure from obstruction to the outflow of bile and resulting stasis is conducive to bacterial growth, a factor in acute cholecystitis.

In 1975 Ternberg and Keating[16] collected reports on 67 infants and children with acute acalculous cholecystitis and added seven of their own. They emphasized that it was frequently a complication of prolonged illness, infection or trauma.

In this series of patients treated in The New York Hospital–Cornell Medical Center, we had two children each between nine and ten years of age, both boys. One, a 9½-year-old child was admitted on 3/29/46. A diagnosis of acute cholecystitis was made and a cholecystectomy was done. There was hemorrhage in the wall of the gallbladder with marked edema and no calculi. The patient ran a febrile course. A blood stream infection due to hemolytic streptococcus was demonstrated. Sixteen days following discharge the patient was readmitted for subacute glomerulonephritis. Fourteen years later (1960) he was well and without evidence of biliary tract disease.

The following is an example of acute acalculous cholecystitis in an individual in whom no specific etiology was evident.

A young boy, age 12, began to experience attacks of severe dull right upper quadrant pain. These recurred and were increasingly frequent over the ensuing three years. Because of persistent symptoms this doctor's son was admitted to the hospital in 1948. A large gallbladder which was not visualized by a Graham series was palpable. Cholecystectomy was performed at which time a diagnosis of acute acalculous cholecystitis was made. The patient has remained symptom-free and is well 30 years later (1978). No anomalies were demonstrated at operation.

The following is an example of acute acalculous cholecystitis in an adult associated with infection.

The patient was admitted on 8/21/45 because of acute abdominal pain, marked tenderness in the right upper quadrant of one day's duration. The temperature was 39.4°, pulse 106, respiration 24. Cholecystectomy was performed for acute acalculous cholecystitis with beginning gangrene. The mucosa was eroded and no calculi were evident. Microscopic examination revealed a gallbladder wall covered by a heavy layer of fibrin filled with areas of hemorrhage. Culture of the gallbladder content revealed salmonella as did the stool cultures. The patient was discharged on 9/6/45. A follow-up report on May 6, 1949 stated that the patient was symptom-free and had recently delivered a normal child.

Munster[9] and associates reported ten cases of acalculous cholecystitis in burned patients. One patient in our series is comparable to the findings in Munster's series.

This 36-year-old man was admitted following a boiler explosion that caused severe burns of the face, neck, chest, both arms and

back. Hemoconcentration was indicated by hemoglobin of 17 g, RBC 6.3, hematocrit 52.2 and WBC 25,000. On the fourteenth hospital day the patient developed symptoms of acute cholecystitis. Cholecystectomy was done and an acalculous gallbladder removed. The patient remained in the hospital for 47 days. There was an uneventful recovery.

Acute acalculous cholecystitis is a relatively infrequent condition encountered in the surgical treatment of biliary tract disease. It occurs in all age groups from very young to very old. In less than half there is a reasonable explanation for its genesis. Untreated it is associated with a high mortality. Treated surgically the mortality rate is higher than any of the several categories of benign biliary tract disease so well reported.

The 139 patients described in this report under the diagnosis of acute acalculous cholecystitis are so variable in history, physical findings and pathological changes in the gallbladder and biliary tract that one may question the value of labeling them as a single entity. Furthermore the diagnosis made at operation may be incorrect because calculi have escaped detection. Then some and in particular those treated primarily by cholecystostomy may later be found to have calcareous material within the biliary tract at subsequent operation. We have offered what seems to be a plausible explanation for these observations but cannot argue that calculi or calcareous material were not present when the primary operation was done. It is because of these variables that the term "acute acalculous cholecystitis" is a somewhat speculative term.

A group of 139 patients diagnosed at operation as having acute acalculous cholecystitis are reported because of the unique problems in management that they present. The gravity of these is reflected by a mortality rate of 6.5% in a hospital with optimum facilities; and that only 78 were accorded a definitive operation. The remaining 61 patients had a compromise procedure because they were considered unable to tolerate the trauma of a cholecystectomy. Two of the 78 whose primary operation was a cholecystectomy required a second operation, whereas those limited to cholecystostomy (61) underwent 41 secondary operations. In 26 instances of the total secondary operations (43), calcareous like material was recovered.

Many of the 139 patients had a recent history of unrelated surgical procedures, major trauma, burns or systemic infection that tended to obscure and delay the diagnosis. The propensity for its development among the injured, the elderly with organ and system impairment and those debilitated by prolonged illness account for the nature and gravity of many of the problems that are associated with surgical treatment. The incidence of these are diminished by early diagnosis and undelayed operation.

Viewed in perspective in relation to the surgical treatment of 11,389 patients for nonmalignant disease of the biliary tract over a period of 45 years (1932–77) is an infrequent condition with a guarded prognosis. Its successful management depends upon the best of surgical judgment and expertise.

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